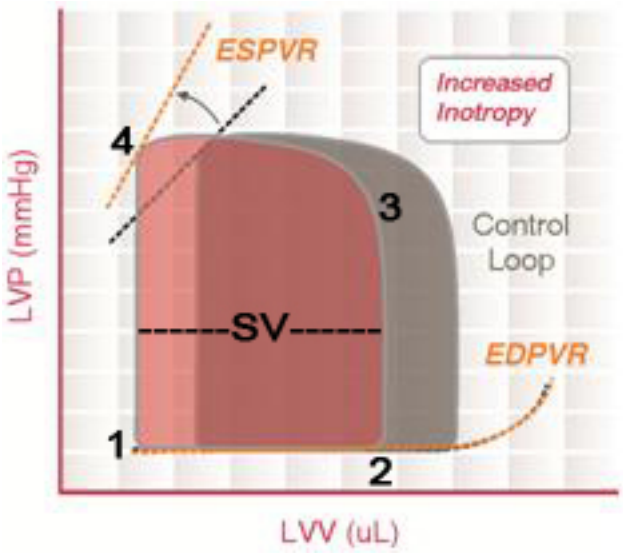
# I. Ventricular PV loops( Tuesday)

**Definition**

**Ventricular Pressure Volume Loop**

This left ventricular pressure volume (P-V) loop shows two loops superimposed on one graph. The grey loop represents the control or normal P-V loop and the pink loop represents a P-V loop with increased inotropy. Because the pink loop is a little easier to visualize, we will use it to demonstrate the various phases and points on a ventricular P-V loop.




1. At point 1, the mitral valve opens as left atrial pressure exceeds left ventricular pressure, and this begins ventricular diastolic filling, lasting from points 1 to 2.

2. Point 2 corresponds to left ventricular end diastolic pressure and volume. At point 2, the left ventricular pressure exceeds left atrial pressure and causes closure of the mitral valve. The phase between points 2 and 3 is known as the isovolumic contraction phase during which the left ventricular pressure increases but left ventricular volume remains unchanged.

3. Point 3 corresponds to opening of the aortic valve and the beginning of ventricular ejection as the left ventricular pressure exceeds aortic diastolic pressure that opens the aortic valve. Phase 3-4 is the ejection phase of ventricular contraction.

4. At point 4, aortic pressure exceeds left ventricular pressure, ventricular ejection ceases and this causes closure of the aortic valve. Point 4 is also known as the end systolic pressure and volume of the left ventricle. The phase from points 4 to 1 is known as isovolumic relaxation, the volume in the left ventricle is constant but the left ventricular pressure is declining.

Stroke volume is the width of the P-V loop, labeled as SV on the loop. It is the difference between end diastolic volume and end systolic volume.

ESPVR stands for end systolic pressure volume relationship and EDPVR stands for end diastolic pressure volume relationship. These curves change based on changes in contractility, afterload and preload. For example, in our loop above, the increased inotropy loop (pink loop) shows a shift in ESPVR upward and to the left (increasing the slope).

EDPVR reflects the diastolic elastance of the left ventricle since change in pressure (P) divided by change in volume (V), $\Delta P / \Delta V$, reflects elastance or stiffness of the ventricle. The elastance increases as the left ventricular volume increases and as volume becomes larger, the relationship becomes a curve (not a straight line). This upward deflection of the curve reflects that the heart becomes difficult to overfill above a certain volume.

# CHF Frank Starl curve: Phenylephrine

**Definition**

The Frank-Starling Curve

With systolic dysfunction, the Frank-Starling curves shifts down and to the right because of the loss of contractility. Stroke volume can be increased by increasing inotropy, decreasing the afterload, or increasing the

preload. Increasing preload will not necessarily increase stroke volume in heart failure patients, and may make pulmonary or systemic congestion worse.

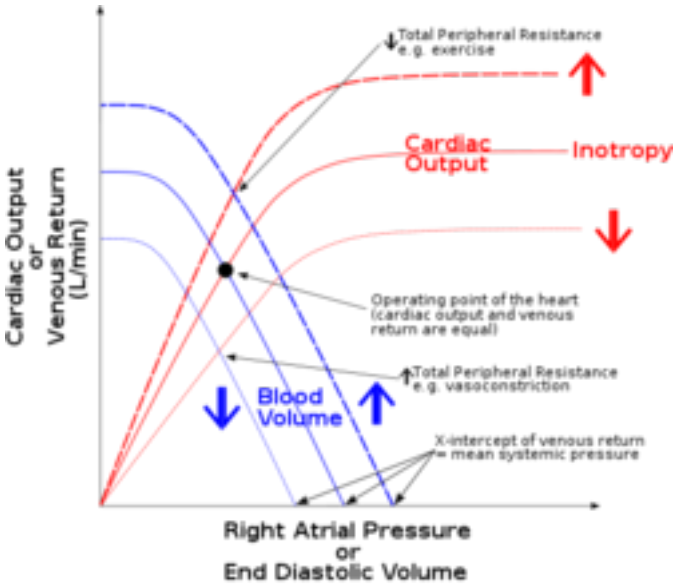
Decreasing afterload with vasodilators can improve stroke volume and ejection fraction because the afterload is often elevated in heart failure and these drugs can increase stroke volume and decrease preload.

Inotropic drugs can increase stroke volume and ejection fraction, and are often used in the treatment of heart failure. Inotropes, like sympathomimetics and phosphodiesterase inhibitors, can increase stroke volume, increase ejection fraction, and reduce preload in the short term. However, prolonged use of inotropic drugs have been shown to increase mortality in some patients, specifically because they can increase oxygen demand.

Phenylepherine is a direct acting sympathomimetic drug that increases venous constriction more than it increases arterial constriction, by acting as an alpha-1 agonist. Acutely, administration of PHE may lead to increase in preload, with increased venous return to the heart, which may (as discussed above) help increase stroke volume somewhat. Importantly, however, this effect is only transient, and over the long run the increase in venous resistance will decrease venous return (and thus preload). Importantly, phenylephrine also leads to an increase in afterload, with increased systemic vasoconstriction. Because alpha1-agonists produce systemic vasoconstriction, the work of the heart increases.

**Whether or not PHE has intrinsic effects on myocardial contractility has in the past been controversial, however the most recent human data (based on infusion directly into the coronary arteries) suggest that PHE does exert mild effects on the myocardium (increased contractility), but that this effect is attenuated in patients with heart failure**. Note that phenylephrine has never been shown to increase cardiac output in any human population.

**Related Media**




## Keyword history

35%/2011

## Sources

*PubMed*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3. J S Landzberg, J D Parker, D F Gauthier, W S Colucci. Effects of myocardial alpha 1-adrenergic receptor stimulation and blockade on contractility in humans. Circulation: 1991, 84(4);1608-14
4. Robert H Thiele, Edward C Nemergut, Carl Lynch. The physiologic implications of isolated alpha(1) adrenergic stimulation. Anesth. Analg.: 2011, 113(2);284-96
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# Lusitropy: PV loop

**Definition**

Lusitropy refers to the ability of the myocardium to relax following excitation contraction coupling. The removal of calcium to the endoplasmic reticulum (SR) promotes relaxation. The sarcoplasmic reticulum calcium ATPase (SERCA), NA/CA exchanger (NCX) and sarcolema CA-ATPase account for 90% removal of calcium. In essence, lusitropy is diastolic function as inotropy (or contractility) is systolic function.

Factors that produce positive lusitropy are: **beta adrenergic agonists** phosphorylate phospholamban via cAMP, reducing calcium available for binding with troponin. Phospholamban is antagonist of SERCA in the non phosphorylated state

Factors that produce negative lusitrophy are: high calcium, impaired sarcoplasmic reticulum calcium ATPase (SERCA). Increase affinity of troponin C, alkalosis that increases calcium sensitivity

Why is this important? Lusitropy along with ventricular hypertrophy, can cause diastolic dysfunction causing increase end diastolic pressure, wedge pressure, PA presure, edema. **The volume loop is smaller, *may* be moved to the left, and *may* be moved up due to decrease compliance.**

# II. Cardiac surface anatomy( Thursday)

**Definition**

**Auscultatory Zones**

Aortic: RUSB (right upper sternal border)

Pulmonic: LUSB (left upper sternal border)

Tricuspid: 4LICS (4th left intercostal space), on the sternal border

Mitral: 5 LICS (5th left intercostal space), several cm lateral to the 4LICS

**Apex Beat**

Located anywhere from the 4th to 5th intercostal space, 6-10 cm lateral to midline

**Borders of the Heart**

Superior: line connecting inferior 2nd L costal cartilage to the superior 3rd R costal cartilage

Inferior: line from the inferior R border of the sternum to the intersection of the 5th intercostal space and the midclavicular line

Right: line drawn from 3rd R costal cartilage to 6th R costal cartilage

Left: line from the L ends of the superior and inferior lines

**ECG Lead Placement**

Note that not all ECG leads have identical sensitivity for ischemia detection. Because not all leads are monitored intraoperatively, judicious selection becomes important

Individual Lead Sensitivity for Detecting Ischemia – V5: 75% – V4: 61%

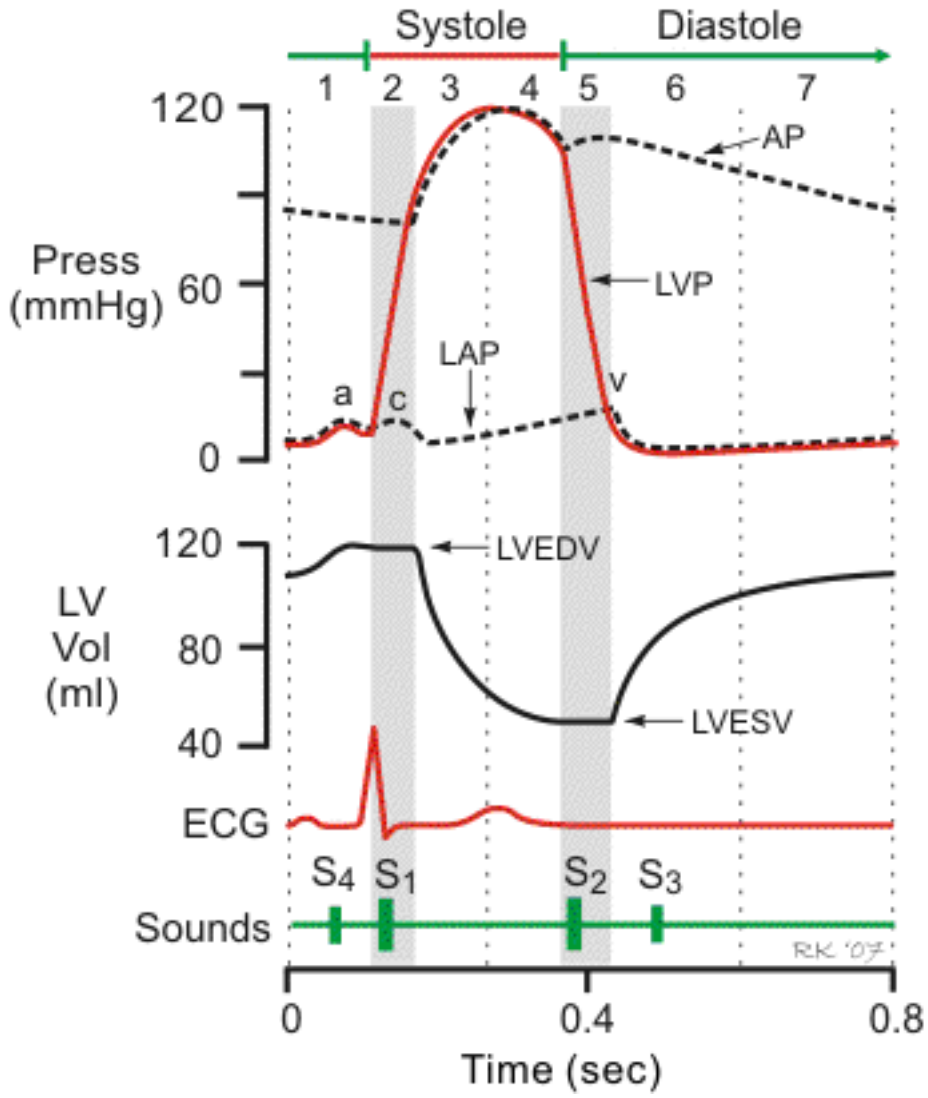
V4 and V5 sensitivity is 90%, which is better than the usual lead II/V5 combination (sensitivity 80%). The **best combination is leads II, V4, and V5**, which has a sensitivity of 98%.

# Cardiac cycle: ECG

Diastole is divided into four phases and comprises 2/3 of the cardiac cycle. It begins when the aortic valve closes. This is the start of Isovolumic Relaxation (volume remains constant but the pressure in the ventricles

fall). The next phase of diastole occurs when the mitral valve opens and allows for rapid ventricular filling. This accounts for the majority of ventricular filling (70-75%). The third phase called diastasis occurs next describing the decrease in passive filling of the ventricles (accounts for 5% of ventricular filling). The final phase is atrial contraction. This typically contributes approximately 20-25% of ventricular filling.

The isovolumic relaxation phase can be used to access diastolic function. This is performed by calculating the instantaneous rate of decline of the LV pressure (-dP/dt) or the time constant of isovolumic decline in the LV pressure (Tau). Many different factors contribute to diastolic function. These include: magnitude of systolic volume, chamber stiffness, elastic recoil of the ventricle, diastolic interaction between the two ventricles, atrial properties and catecholamines. All of these interactions can impair the ability of the heart to fill.




**Keyword history**

- 56%/2015
- 12%/2010
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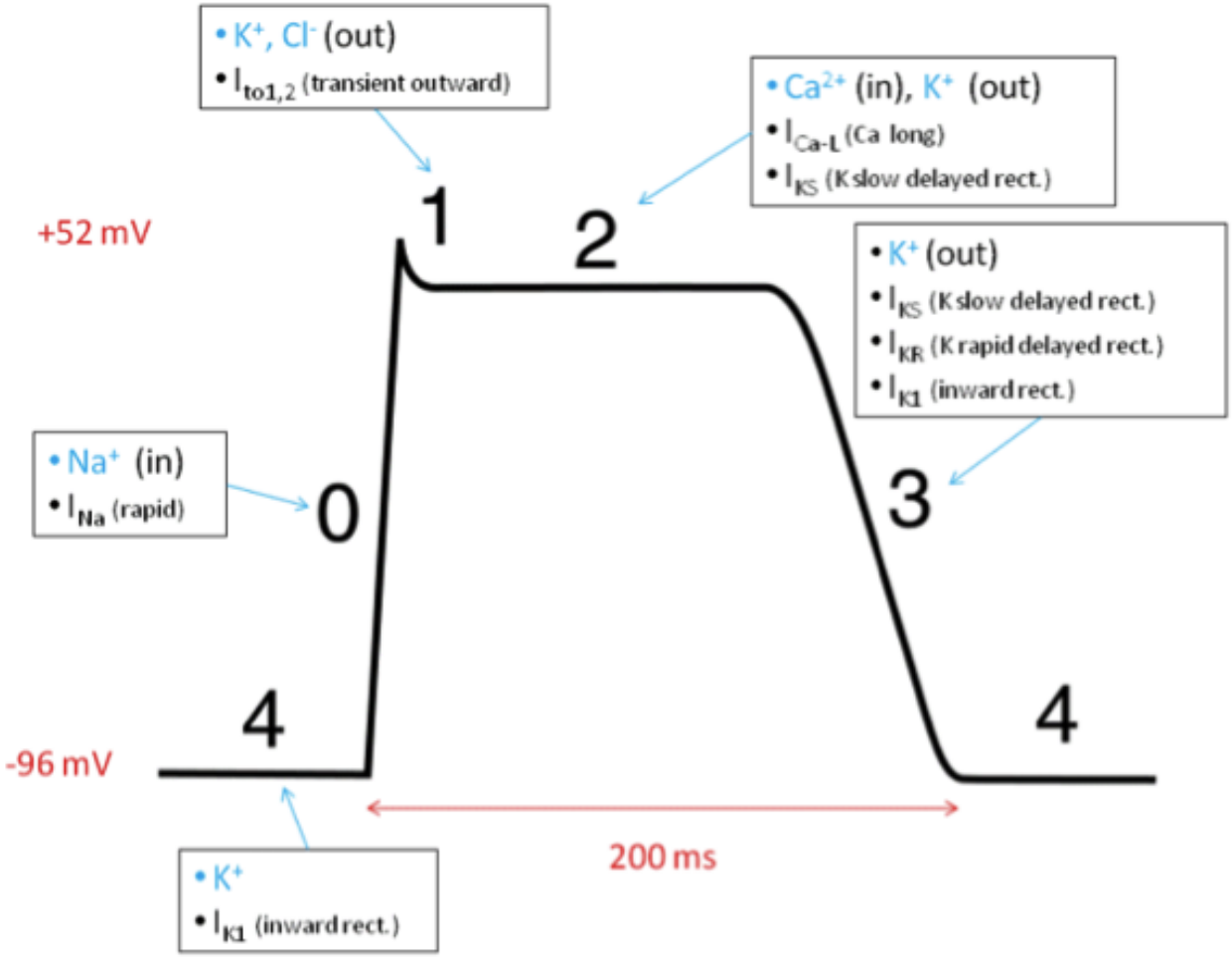
# III. Myocyte repolarization: Ionic flow( Friday)

For skeletal muscle, Na+ mediated depolarization (Phase 1) is quickly followed by repolarization (Phase 3). Repolarization is mediated by closure of the Na+ channels responsible for depolarization, and opening of

voltage-gated K+ rectifier channels, which allows for potassium efflux. These K+ channels will remain open and hyperpolarize the cell membrane is restored to its resting state (-90 mV).

Cardiac myocytes are more complicated. Depolarization is initially followed by a “plateau phase” (Phase 2) where the membrane potential remains positive for 0.2-0.3 seconds. During this phase, Ca2+ influx through L-type Ca2+ channels balances K+ efflux through slow delayed rectifier K+ channels. Eventually, the Ca2+ channels close while the K+ permeability increases as rapid rectifier K+ channels open, allowing for rapid repolarization (Phase 3). K+ channels remain open until the resting membrane potential is restored.




**Cardiac Myocyte, Image Source: Wikimedia Commons**

# Myocardial O2 consumption: determinants

**Definition**

The amount of oxygen consumed is determined by the basal O2 consumption, wall tension, contractility and heart rate.

**Wall tension =(pressure*radius)/2*wall thickness**

pressure = afterload; radius = preload

Pressure alone is not a good measure for myocardial O2 consumption unless the other factors above are considered.

The greater the contractility, the more oxygen the myocardium consumes. Faster, more powerful contractions (increased dP/dT) requires more energy.

Increased HR leads to increased myocardial O2 consumption. Heart rate is extremely important in that it influences not only supply, but demand as well, as the myocardium is perfused during diastole.

In the unanesthetized individual, myocardial consumption is the main determinant of myocardial supply.

# Arterial waveform: Peripheral vs. Central

As the arterial pressure wave travels from the central aorta to the periphery, the arterial upstroke becomes steeper, the systolic peak becomes higher, the dicrotic notch appears later, the diastolic wave becomes more prominent, and end-diastolic pressure becomes lower. **Thus, when compared with central aortic pressure, peripheral**

**arterial waveforms have higher systolic pressure, lower diastolic pressure, and wider pulse pressure**. Furthermore, there is a delay in arrival of the pressure pulse at peripheral sites, so the systolic pressure upstroke begins approximately 60 msec later in the radial artery than in the aorta. Despite morphologic and temporal differences between peripheral and central arterial waveforms, **MAP in the aorta is just slightly greater than MAP in the radial artery.**